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Effect of chronic antigen inhalation in guinea pigs.

Yamada N, Funayama K, Ono Y.

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To examine the role of airway inflammation in airway hyperresponsiveness (AHR), we examined the effect of chronic antigen inhalation in sensitized guinea pigs. Guinea pigs were actively sensitized with dinitrophenylated Ascaris suum extract (DNP-As) and repeatedly exposed to aerosolized DNP-As antigen once a day for 4 or 10 days. Twenty-four hours after the last antigen exposure, airway responsiveness to inhaled acetylcholine (ACh) and bronchoalveolar lavage (BAL) were studied. The guinea pigs receiving 4 days of exposure to antigen demonstrated an increase in airway responsiveness to inhaled ACh ($p < 0.05$). On the other hand, the guinea pigs receiving 10 days of exposure to antigen showed no significant change in airway responsiveness to inhaled ACh. BAL fluid analysis indicated that a significant increase in the number of eosinophils and neutrophils was observed in both groups of guinea pigs. A significant increase in the number of lymphocytes in BAL fluid was observed in guinea pigs exposed for 10 days, but not in those exposed for 4 days. We conclude that repeated exposure to antigen induced both development and suppression of AHR. Our results suggest that airway inflammation may play a role in both the development and suppression of AHR.

PMID: 9513585 [PubMed - indexed for MEDLINE]

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Development of a prolonged eosinophil-rich inflammatory leukocyte infiltration in the guinea-pig asthmatic response to ovalbumin inhalation.

Dunn CJ, Elliott GA, Oostveen JA, Richards IM.

Department of Hypersensitivity Diseases Research, Upjohn Company, Kalamazoo, Michigan.

Considerable attention has recently focused on the role of inflammation in the pathophysiology of asthma, with special emphasis on "late-phase" bronchoconstriction and increased airway hyperreactivity after antigen challenge in sensitized subjects. The present report describes the histopathologic changes in guinea-pig lung and trachea at various time intervals after ovalbumin inhalation in nonsensitized (control) and sensitized animals. Bronchoalveolar lavage (BAL) was also used to assess the accompanying accumulation of intraluminal leukocytes. A distinct leukocyte margination, consisting of neutrophils and eosinophils, was observed in the peribronchial vasculature as early as 8 min postchallenge in sensitized guinea pigs. At 6 h, the eosinophils predominated and migrated to the peribronchiolar smooth muscle layer. Between 6 h and 18 h, eosinophils were seen in tracts between the smooth muscle cell layers, accumulating in large numbers in the bronchial mucosal epithelium. This pattern persisted for at least 7 days postchallenge during which eosinophils remained the dominant cell type present. Peribronchiolar accumulation of neutrophils and mononuclear cells was minimal at all time points studied. Intraluminal mucus eosinophilia developed between 18 h and 7 days. A similar pattern of eosinophil infiltration was observed in the tracheal epithelium. Control, nonsensitized, guinea-pig lungs showed minor changes with little or no eosinophil infiltration at any time after antigen challenge. These findings correlated well with the BAL study in which sensitized guinea pigs exhibited a marked delayed increase in eosinophil counts between 18 h and 7 days compared with that in nonsensitized animals.(ABSTRACT TRUNCATED AT 250 WORDS)

PMID: 3345036 [PubMed - indexed for MEDLINE]

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Bronchial inflammation and hyperreactivity after anaphylactic shock in guinea pigs actively sensitized by systemic or aerosol route.

Tarayre JP, Aliaga M, Barbara M, Malfetes N, Vieu S, Tisne-Versailles J.

Centre de Recherche Pierre Fabre, Castres, France

Hyperreactivity and bronchial inflammation resulting from active anaphylactic shock induced by aerosol have been studied in guinea pigs after sensitization by intramuscular injection of large-dose ovalbumin or aerosol ovalbumin. When animals were sensitized by i.m. injection of 30 mg/kg ovalbumin, hyperreactivity to inhalation of histamine was obtained 1-3 h after shock. In bronchoalveolar lavage (BAL) fluid an increase in the number of eosinophils (6-48 h after shock) and neutrophils (6-24 h) was observed. When guinea pigs were sensitized by aerosol route, the hyperreactivity to histamine inhalation appeared 1-6 h after shock. In BAL fluid the number of mononuclear cells dropped (1-3 h) and then increased (24-48 h); the number of neutrophils (6-48 h) and eosinophils (24-48 h) increased. The results observed during these two types of sensitization were compared to those obtained after sensitization by injection of a large dose of ovalbumin mixed with Freund's complete adjuvant.

PMID 2072813 [PubMed - indexed for MEDLINE]

1: Int Arch Allergy Immunol 1995 Sep;108(1):60-7 Related Articles, Links

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Induction of leucocyte recruitment and bronchial hyperresponsiveness in the guinea pig by aerosol administration of interleukin-2.

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Exposure of guinea pigs to an aerosol of human recombinant interleukin-2 (IL-2; 30 micrograms) resulted in an increase in the numbers of eosinophils and macrophages recovered from bronchoalveolar lavage fluid (BALF) 24 h later. This was accompanied by a bronchial hyperresponsiveness to intravenous acetylcholine. In guinea pigs sensitized to ovalbumin, exposure to IL-2 caused an increase in the number of macrophages, but not eosinophils in BALF and bronchial hyperresponsiveness to acetylcholine did not develop. In guinea pig skin, intradermal injection of IL-2 (10(-14) to 10(-9) mol/site) had no effect on 111In-eosinophil accumulation, measured over 3 h, suggesting that IL-2 does not act directly to recruit eosinophils. The hypothesis that IL-2 may be acting via release of interleukin-5 (IL-5) was tested using an antibody to IL-5 (TRFK-5; 1 mg/kg). Treatment with TRFK-5 1 h before exposure to IL-2 aerosol had no effect on the numbers of macrophages or eosinophils recovered from BALF 24 h later, although there was a tendency for reduced bronchial hyperresponsiveness to acetylcholine. These results suggest that (1) IL-2 is not a directly acting chemoattractant for eosinophils in the guinea pig, (2) the action of IL-2 to increase bronchial hyperresponsiveness is also indirect, partly via generation of IL-5, and (3) immunological sensitization alters the response of both eosinophils and bronchial smooth muscle to IL-2.

PMID: 7647587 [PubMed - indexed for MEDLINE]